

Conclusion: patients with mild DC exhibit a diffusely blunted MBF reserve in comparison with normal controls; in DC segments the contractile response to low dose dobutamine is unrelated to regional coronary flow response to dipyridamole.

1206-145 Quantitation of Infarct Size in Rat Myocardium Using F-18 Deoxyglucose and a New High-Resolution microPET System

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Background: PET imaging of small animals may allow the study of transgenic disease models or facilitate the evaluation of new radiopharmaceuticals. Therefore, we explored the potential of a new high-resolution (~2 mm) microPET system for imaging the myocardium in 16 male rats weighing 344 ± 42 g.

Methods: The left coronary artery was ligated in 8 rats and 8 rats were sham operated. After 1.5 hrs, F-18 deoxyglucose (FDG; 1.5 mCi) was injected iv; 2.5 hrs later, 3-D data were acquired for 30 min. In each rat, 4 to 6 LV short-axis cuts were reconstructed with filtered backprojection. Relative threshold counts for the anterior and inferior LV circumference were derived from the control rats and applied to the infarct rats. The ratio of the total tracer uptake defect area to the LV cross-sectional area R_{PET} was determined in each infarct animal. The heart was removed, sliced into 4 to 6 sections and stained with TTC.

Results: The total infarct-to-LV ratio R_{TTC} on digital planimetry varied from 2 to 54%. R_{PET} correlated linearly with R_{TTC} ($r = 0.95$, $p < 0.001$) but tended to underestimate infarct size. The two smallest infarcts (2% and 6%) were seen only on the TTC sections but not on PET.

Conclusion: High-resolution microPET affords the noninvasive visualization of regional myocardial tracer uptake and uptake defects in only 11 mm large hearts with a 2 mm thick LV wall. Thus, microPET allows use of small laboratory animals for studying for example transgenic rat models or new radiopharmaceuticals at a low cost.

1206-146 Clinical Significance of Increased Fluorine-18 Deoxyglucose (FDG) Uptake in Normoperfused Myocardium Without Ischemic Insult Under Fasting Condition

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Purpose and Methods: Increased uptake of FDG in hypoperfused area is considered to indicate ischemic but viable myocardium. However, increased FDG uptakes are also observed in normoperfused myocardium without ischemic injury (normal myocardium) under fasting condition. To evaluate the clinical significance of increased FDG uptakes in normal myocardium under fasting condition, we performed nitrogen-13 ammonia (NH_3) and FDG positron emission tomography (PET) in 66 consecutive patients with ischemic heart disease (42 were myocardial infarction (MI), 19 were variant angina (VA) and 5 were effort angina (EA)); 51 men). Patients with overt diabetes were excluded. When PET images were analyzed excluding the infarct-related area, the territory of coronary arteries having significant stenosis and showing vasospasm, 16 (group A) showed increased FDG uptakes in the area of normal NH_3 uptakes and 50 (group B) did not. The upper limit of normal FDG uptake was defined as the mean + 2SD of normal data in our institution.

Results: The serum glucose levels (98 ± 10 mg/dl vs 98 ± 12 mg/dl) and insulin levels (7.6 ± 5.5 IU/l vs 8.1 ± 3.9 IU/l) were not different between two groups. The serum free fatty acid levels were lower in group A than in group B (0.47 ± 0.16 mEq/l vs 0.62 ± 0.28 mEq/l, $p < 0.05$), but the values were within normal range (0.50 ± 0.35 mEq/l). The distribution of clinical diagnosis was not different between 2 groups (MI: 9, VA: 6, EA: 1 vs MI: 33, VA: 13, EA: 4). When patients were re-classified into "Active-group" (within 2 weeks after onset of MI and the phase of unstable angina) and "Inactive-group", 10 of 16 group A patients (63%) were included in Active-group, but only 5 of 50 group B patients (10%, $p < 0.01$).

Conclusions: Our data indicate that an increase in FDG uptake in normal myocardium are closely related to contiguous phase of critical myocardial ischemic events, suggesting that myocardial ischemic events may produce acceleration of glucose utilization even in remote normal myocardium.

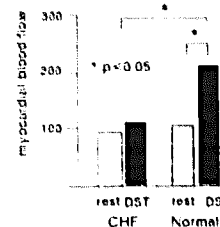
1206-147 Impairment of Myocardial Blood Flow Reserve, in Patients With Coronary Artery Disease and Heart Failure, Depends on Severity of Left Ventricular Dysfunction and Not Severity of Coronary Stenosis

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Background: Myocardial blood flow reserve (MFR) is impaired in patients with coronary artery disease (CAD), not only in arteries with significant (>70%), but also non-significant lesions. We examined whether this MFR-impairment would be related to the degree of LV dysfunction.

Methods: We studied 12 pts with CAD and congestive heart failure (CHF) (LVEF 0.34 ± 0.02 , age 63 ± 3) and compared them with 12 age and sex-matched controls with similar CAD (LVEF 0.56 ± 0.03 , age 64 ± 4). Medication was withheld pre-study, depending on the plasma half time. MFR was assessed with positron emission tomography and N-13 ammonia at rest and after infusion of dipyridamole, in the non-infarcted related artery (non-IRA).

Results: In stenotic non-IRA coronary arteries MFR was impaired in both groups ($p = NS$).



In contrast, in the non-stenotic (<70% stenosis) non-IRA coronary arteries, MFR was impaired during dipyridamole stress test (DST) in CHF (fig). Further, in CHF, patients MFR showed a relation ($r = 0.6$, $p < 0.05$) with LVEF, but not with the severity of CAD.

Conclusion: In patients with CHF with underlying CAD and non-stenotic coronary arteries, myocardial blood flow at rest was similar, but MFR was impaired, which correlated with LVEF. The impairment of flow reserve may play an important role in the pathogenesis of ischemic heart failure.

1206-148 Prospective Assessment of Left Ventricular Adaptation With Electron Beam Computed Tomography (EBT) in High vs. Low Exercise Rehabilitation After First Anterior Myocardial Infarction

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High level (Phase II) exercise in patients (pts.) with large myocardial infarctions (MI) has been controversial. This prospective trial was designed to quantitatively assess cardiac size, function and infarct size in 30 pts. (5 women) randomized to regular (Phase I & II, "high") or "low" exercise (no increase in VO_2 uptake) after first Q-wave MI. All pts. underwent supervised training sessions for six months without knowledge of their group assignment and received β -blockers and ACE-inhibitors. The infarct-related vessels were patent. EBT and sestamibi scans were performed at discharge (5-10 days after MI, "base"), and 6 months to determine LV and RV enddiastolic (EDV), endsystolic (ESV), and stroke volumes (SV), as well as LVEF. LV muscle mass (MM), and infarct size. The two groups did not differ in age (56 ± 12 vs 56 ± 12 yrs), infarct size (34 ± 25 vs $29 \pm 22\%$ of LV) or number of significant stenoses. Results are shown below:

	High 6 months	% from base	Low 6 months	% from base
LVEDV [ml]	215 ± 57	14 ± 42	190 ± 46	2 ± 27
LVESV [ml]	119 ± 48	9 ± 35	102 ± 43	0 ± 28
LVSF [ml]	96 ± 23	5 ± 17	87 ± 13	2 ± 22
LVEF [%]	46 ± 9	0 ± 7	48 ± 10	0 ± 11
RVFEDV [ml]	193 ± 43	14 ± 34	180 ± 31	7 ± 23
FVESV [ml]	106 ± 31	11 ± 29	90 ± 25	5 ± 17
MM [g]	179 ± 40	-3 ± 39	156 ± 30	17 ± 26

Conclusion: 1) In patients with moderately large anterior MIs, treated with ACE inhibition, there is no significant change in cardiac function, volume, and muscle mass from baseline to six months. 2) There appears to be no detrimental effect of high level rehabilitation exercise on cardiac size and function parameters.